PPARα is essential for microparticle-induced differentiation of mouse bone marrow-derived endothelial progenitor cells and angiogenesis

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Résumé en anglais	**Background** Bone marrow-derived endothelial progenitor cells (EPCs) are critical for neovascularization. We hypothesized that microparticles (MPs), small fragments generated from the plasma membrane, can activate angiogenic programming of EPCs. **Methodology/Principal Findings** We studied the effects of MPs obtained from wild type (MPsPPARα+/+) and knock-out (MPsPPARα−/−) mice on EPC differentiation and angiogenesis. Bone marrow-derived cells were isolated from WT or KO mice and were cultured in the presence of MPsPPARα+/+ or MPsPPARα−/− obtained from blood of mice. Only MPsPPARα+/+ harboring PPARα significantly increased EPC, but not monocytic, differentiation. Bone marrow-derived cells treated with MPsPPARα+/+ displayed increased expression of pro-angiogenic genes and increased in vivo angiogenesis. MPsPPARα+/+ increased capillary-like tube formation of endothelial cells that was associated with enhanced expressions of endothelial cell-specific markers. Finally, the effects of MPsPPARα+/+ were mediated by NF-κB-dependent mechanisms. **Conclusions/Significance** Our results underscore the obligatory role of PPARα carried by MPs for EPC differentiation and angiogenesis. PPARα-NF-κB-Akt pathways may play a pivotal stimulatory role for neovascularization, which may, at least in part, be mediated by bone marrow-derived EPCs. Improvement of EPC differentiation may represent a useful strategy during reparative neovascularization.
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